

The Effect of Coronavirus on the Liver and Histopathological Findings

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Letter to the Editor

Coronavirus is a single-stranded large RNA virus that infects humans (1). According to the World Health Organization (WHO), the cause of SARS-CoV-2 is called disease COVID-19. Similar to SARS-CoV, SARS-Cov-2 mainly attacks the respiratory system. Symptomatic conditions indicate clinical symptoms of the disease, symptoms of fever, cough, fatigue and other respiratory infections (2). Although the main clinical findings of COVID-19 are associated with lung injury, which is the main cause of mortality, findings related to the involvement of other organs such as cardiac and liver have also been reported (3).

No evidence of isolated acute liver failure has been observed in COVID-19 patients, but there are articles on autopsy cases on histopathological data of liver damage during the disease. Zhang Y and colleagues from Wuhan University Zhongnan Hospital reported that they did not determine a definite change in the macroscopic appearance of the liver in the autopsy results of COVID-19 related deaths (1). In histopathological analysis, they stated that there was mild sinusoidal dilatation and minimal lymphocytic infiltration and no other special damage was observed (1).

Li Y et al. also found that mild sinusoidal lymphocytic infiltration and sinusoidal dilatation were the main pathological findings in histopathological examination of the liver. There were also mild steatosis and multifocal hepatic necrosis in some patients. In addition, hyperinflammatory reactions were associated

with COVID-19 and COVID-19 may contribute to liver damage in pre-existing chronic liver disease (4).

In addition to other studies, Xu Z et al. reported that portal inflammation was not prominent. They observed mild lobular and portal activity, moderate microvesicular steatosis (5). Tian S et al. also observed hepatic necrosis foci in the liver zone 1 and zone 3 areas. They reported that they did not find out any serious inflammatory cell infiltration, cytoplasmic balloon degeneration, mallory hyaline or fibrosis. They emphasized that the current findings were consistent with the pattern of acute liver injury, and no more serious histological changes such as coagulative necrosis and severe cholestasis occurred (6). Yao et al. reported that they rarely observed canalicular cholestasis (7).

Chai X et al. stated that angiotensin converting enzyme 2 (ACE2), which is the default receptor for SARS-CoV-2, is expressed more intensely in bile duct epithelial cells, can be directly attached to these cells except hepatocytes. For this reason, they thought that the liver abnormalities of the patients might be caused by cholangiocyte dysfunction, not hepatocyte damage (8). They emphasized that sinusoidal dilatation was due to cardiogenic venous outflow slowing, however, other histological described findings were probably related to the patient's primary disease, namely COVID-19. Drugs used in the treatment of COVID-19 (hydroxychloroquine and azithromycin) can also cause liver injury.

These drugs have been shown to cause various degrees of hepatotoxicity. Also, the hypoxic condition commonly associated with COVID-19 pneumonia may make hepatocytes more susceptible to toxic injuries (6). Decreased perfusion to the liver due to heart failure in these patients may also exacerbate this process (4). These studies in the literature show that during the clinical course of COVID-19, cell injury due to direct viral origin and potential hepatotoxicity caused by therapeutic drugs occur. Especially in patients with serious or critical disease, liver damage has been observed to be significant. The underlying mechanisms of hepatic injury can be multifactorial and may vary individually. There are many factors related to this condition, including direct viral attack, hepatotoxicity of therapeutic drugs, hyper-inflammatory reactions, pre-existing chronic liver disease and hypoxemic state (4). As a result, in patients with COVID-19, mild increase in sinusoidal lymphocytic infiltration, sinusoidal dilatation, mild steatosis and multifocal hepatic necrosis are the main histopathological abnormalities. Liver failure is not a prominent feature of the disease (9). Therefore, biopsy in this process is not recommended as it may cause greater harm than possible benefit.

References

1. Zhang Y, Zheng L, Liu L, Zhao M, Xiao J, Zhao Q. Liver impairment in COVID-19 patients: A retrospective analysis of 115 cases from a single centre in Wuhan city, China. *Liver International* **2020**.
2. Chen N, Zhou M, Dong X, Qu J, Gong F, Han Y, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. *Lancet* **2020**; 395(10223): 507-513.
3. Zhu N, Zhang D, Wang W, Li X, Yang B, Song J, et al. A novel coronavirus from patients with pneumonia in China, 2019. *New Engl J Med* **2020**.
4. Li Y, Xiao SY. Hepatic involvement in COVID-19 patients: pathology, pathogenesis and clinical implications. *J Medical Virol* **2020**.
5. Xu Z, Shi L, Wang Y, Zhang J, Huang L, Zhang C, et al. Pathological findings of COVID-19 associated with acute respiratory distress syndrome. *Lancet Resp Med* **2020**; 8(4): 420-422.
6. Tian S, Xiong Y, Liu H, Niu L, Guo J, Liao M, et al. Pathological study of the 2019 novel coronavirus disease (COVID-19) through postmortem core biopsies. *Modern Pathol* **2020**: 1-8.
7. Yao X, Li T, He Z, Ping Y, Liu H, et al. A pathological report of three COVID-19 cases by minimally invasive autopsies. *Zhonghua bing li xue za zhi= Chi J Pathol* **2020**; 49: 009
8. Chai X, Hu L, Zhang Y, Han W, Lu Z, Ke A, et al. Specific ACE2 expression in cholangiocytes may cause liver damage after 2019-nCoV infection. *BioRxiv* **2020**.
9. Humar A, McGilvray I, Phillips MJ, Levy GA. Severe acute respiratory syndrome and the liver. *Hepatology (Baltimore, Md)* **2004**; 39(2): 291.

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